

09/419,901
updated Search
L/Cook 6/16/05-

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(FILE 'HOME' ENTERED AT 16:21:05 ON 16 JUN 2005)

FILE 'STNGUIDE' ENTERED AT 16:21:13 ON 16 JUN 2005

FILE 'HOME' ENTERED AT 16:21:22 ON 16 JUN 2005

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
16:21:43 ON 16 JUN 2005

L1	304 S (MYOFILAMENT PROTEIN)
L2	159 S (CHEMICAL ADDUCT)
L3	1 S L1 AND L2
L4	180 S L1 AND TROPONIN?
L5	95 S L1 AND ACTIN?
L6	12 S L1 AND DESMIN?
L7	110 S L1 AND MYOSIN?
L8	1 S L4 AND ADDUCT?
L9	1 S L5 AND ADDUCT?
L10	1 S L6 AND ADDUCT?
L11	101 S L4 AND MUSCLE?
L12	1 S L2 AND TROPONIN?
L13	15 S L2 AND REVIEW?
L14	10 DUPLICATE REMOVE L13 (5 DUPLICATES REMOVED)
L15	14 S L2 AND ACTIN?
L16	1 S L2 AND MYOSIN?
L17	1 S L2 AND DESMIN?
L18	5 DUPLICATE REMOVE L15 (9 DUPLICATES REMOVED)
L19	62 S L5 AND MUSCLE?
L20	30 DUPLICATE REMOVE L19 (32 DUPLICATES REMOVED)
L21	0 S L20 AND TRANSLATION?
L22	11 S L1 AND TRANSLATION?
L23	0 S L22 AND ADDUCT?
L24	1 S L1 AND ADDUCT?
L25	7 DUPLICATE REMOVE L22 (4 DUPLICATES REMOVED)

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DUPLICATE 1

AN 1994:187066 BIOSIS

DN PREV199497200066

TI Detection of reduced acetaldehyde protein adducts using a unique monoclonal antibody.

AU Klassen, Lynell W. [Reprint author]; Tuma, Dean J.; Sorrell, Michael F.; McDonald, Thomas L.; Devasure, Jane M.; Thiele, Geoffrey M.

CS University Nebraska Medical Center, Dep. Internal Medicine, 600 S. 42nd St., Omaha, NE 68198-3332, USA

SO Alcoholism Clinical and Experimental Research, (1994) Vol. 18, No. 1, pp. 164-171.

CODEN: ACRSDM. ISSN: 0145-6008.

DT Article

LA English

ED Entered STN: 26 Apr 1994

Last Updated on STN: 27 Apr 1994

AB Acetaldehyde (AA), the major product of alcohol metabolism, has been shown to bind to proteins *in vivo* and form **chemical adducts**.

These AA-protein adducts have been shown to alter protein structure and function and may result in tissue damage. Recent reports have shown that polyclonal antibodies can be produced that recognize proteins modified *in vitro* with AA in the presence of sodium cyanoborohydride (NaCNBH-3), a strong reducing (R) agent. Antibodies prepared in this way have been shown to recognize proteins in the livers of rats fed alcohol chronically. Because multiple AA-protein adducts can be recognized by polyclonal antisera, and a variety of adducts may be formed *in vitro* or *in vivo*, this study was designed to develop monoclonal antibodies specific for proteins modified by AA. In addition, adducts formed under R conditions are probably chemically different than those formed under nonreducing (NR) conditions, and monoclonal antibodies may provide the specificity required to distinguish these chemical differences. Balb/c mice were immunized with bovine brain tubulin that was modified by treatment with 5 mM AA for 7 days under NR conditions. Sera from immunized animals were tested for antibody activity to the immunogen (protein-NR) and for cross-reactivity to protein-R and unmodified protein. Although the highest serum antibody titers were seen toward the NR adduct, antibodies to the R adduct were also detected. This activity difference was independent of the carrier protein, because NR and R bovine serum albumin, keyhole limpet hemocyanin, and **actin** also gave similar results when used as the adducted protein. Surprisingly, all the monoclonal antibodies (RT1.1, RT1.2, RT1.3, and RT1.4) produced by hybridomas generated from spleen cells from NR-tubulin immunized mice recognized the R and not the NR adduct. One of these hybridomas (RT1.1) produces an IgG2b antibody that reacts with all tested proteins that have been modified with AA under chemical R conditions. Because of its monoclonal specificity, this antibody may be useful in probing for the presence of R AA-protein adducts made both *in vitro* and *in vivo*.

CC Biochemistry methods - General 10050

Biochemistry studies - General 10060

Enzymes - Methods 10804

Metabolism - General metabolism and metabolic pathways 13002

Toxicology - General and methods 22501

Immunology - General and methods 34502

IT Major Concepts

Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics); Immune System (Chemical Coordination and Homeostasis); Metabolism; Toxicology

IT Chemicals & Biochemicals

ACETALDEHYDE; ALCOHOL

IT Miscellaneous Descriptors

ALCOHOL; ANALYTICAL METHOD; ELISA; TOXICOKINETICS

ORGN Classifier

Bovidae 85715

Super Taxa

Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

bovine

Taxa Notes

Animals, Artiodactyls, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Vertebrates

ORGN Classifier

Leporidae 86040

Super Taxa

Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

rabbit

Taxa Notes

Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman
Mammals, Vertebrates

RN 75-07-0 (ACETALDEHYDE)

64-17-5 (ALCOHOL)

ANSWER 2 OF 5 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
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Metabolism - General metabolism and metabolic pathways 13002

Toxicology - General and methods 22501

Immunology - General and methods 34502

IT Major Concepts

Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and
Molecular Biophysics); Immune System (Chemical Coordination and
Homeostasis); Metabolism; Toxicology

IT Chemicals & Biochemicals

ACETALDEHYDE; ALCOHOL

IT Miscellaneous Descriptors

ALCOHOL; ANALYTICAL METHOD; ELISA; TOXICOKINETICS

ORGN Classifier

Bovidae 85715

Super Taxa

Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

bovine

Taxa Notes

Animals, Artiodactyls, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Vertebrates

ORGN Classifier

Leporidae 86040

Super Taxa

Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

rabbit

Taxa Notes

Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman
Mammals, Vertebrates

RN 75-07-0 (ACETALDEHYDE)

64-17-5 (ALCOHOL)

on STN
 AN 86145956 EMBASE
 DN 1986145956
 TI Thin **myofilament proteins** in norm and heart failure.
 I. Polymerizability of myocardial Straub **actin** in acute and chronic heart failure.
 AU Karsanov N.V.; Pirtskhalaishvili M.P.; Semerikova V.J.; Losaberidze Sh. N.
 CS The Republican Centre of Medical Biophysics of the Georgian SSR Public Health Ministry, 380092 Tbilisi, Russia
 SO Basic Research in Cardiology, (1986) Vol. 81, No. 2, pp. 199-212.
 CODEN: BRCAB7
 CY Germany
 DT Journal
 FS 018 Cardiovascular Diseases and Cardiovascular Surgery
 029 Clinical Biochemistry
 LA English
 ED Entered STN: 911210
 Last Updated on STN: 911210
 AB The reduced and intrinsic viscosities of myocardial Straub F-**actin** from the left ventricle of a practically healthy man were equal to 3.05 ± 0.2 and 2.4 ± 0.32 and from the right ventricle were 2.37 ± 0.2 and 2.1 ± 0.3 dl/g, respectively (the difference between ventricles was not significant). The average length of filaments measured by flow birefringence technique was equal to 1.3 ± 0.04 μ m, the number-average length ($\langle L \rangle_n$), determined by the electron microscopy was 1.4 μ m, the weight-average length ($\langle L \rangle_w$), was 2 μ m and the maximal one was 5.5 μ m. The histograms showed that the most characteristic length was that of 0.8-1.2 μ m. According to the flow birefringence data canine myocardial F-**actin** had a length similar to that of myocardial F-**actin** from a practically healthy man, though its reduced and intrinsic viscosities were higher. In acute and especially chronic congestive heart failure the **actin** polymerizability was sharply reduced. In consequence, in acute heart failure the number-average length of F-**actin** filaments was decreased by 43% and in congestive heart failure by 65.7%. The characteristic length in acute heart failure shifts to the range of 0.2-0.6 μ m, while in congestive heart failure the range is 0.2-0.4 μ m. This fact can possibly explain why during preparation of **actin** from the pathologically changed myocardium according to the methods including purification by the cycles of polymerization-sedimentation-depolymerization, the pathologically changed **actin** is discarded and the normal **actin** remains. A definite parallel was observed between the reduction of **actin** polymerizability and the ability of myocardial glucerinated fiber bundles (MBGF) to generate force. We conclude that the changes of **actin** properties in heart failure may cause a decrease in contractibility of the myocardial contractile protein system.
 CT Medical Descriptors:
 *heart failure
 *heart muscle cell
 *myofilament
 dog
 electron microscopy
 human
 priority journal
 etiology
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 clinical article
 animal experiment
 nonhuman
 heart
 muscle
 Drug Descriptors:

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 Drug Descriptors:

AN 1993:101018 BIOSIS
DN PREV199395056214
TI Estrogen-induced morphological and immunohistochemical changes in stroma and epithelium of rat ventral prostate.
AU Zhao, G. W.; Holterhus, P. M.; Dammshaeuser, I.; Hoffbauer, G.; Aumüller, G. [Reprint author]
CS Dep. Anat. Cell Biol., Robert-Koch-Str., D-3550 Marburg, Germany
SO Prostate, (1992) Vol. 21, No. 3, pp. 183-199.
CODEN: PRSTDS. ISSN: 0270-4137.
DT Article
LA English
ED Entered STN: 9 Feb 1993
Last Updated on STN: 10 Feb 1993
AB Prostatic smooth **muscle** cells have been regarded to play a major pathogenetic role during the development of benign prostatic hyperplasia (BPH) in elderly men. Altered hormonal signals (increased estrogen) have been made responsible for the "metabolic" transformation of prostatic smooth **muscle** cells, which were thought to produce increased amounts of connective tissue fibers observed in BPH. In order to find out the role of metabolically "activated" smooth **muscle** cells, hormone stimulation experiments were performed in male rats. The effects of androgen deprivation and estrogen stimulation were recorded by semiquantitative analysis of intermediate and **myofilament proteins** in stromal smooth **muscle** cells. In castrated or estrogen-treated or estrogen-treated and castrated animals, the reduction of the glandular lumen is the most obvious morphological alteration, accompanied by an increase in connective tissue. Regressive changes occurred most rapidly in castrated animals (already within the first week), slower in castrated estrogen-treated animals and still slower in normal estrogen-treated animals. Regression of the epithelium was accompanied by a marked decrease in immunoreactivity for prostatic binding protein (PBP) in castrated animals, while PBP immunoreactivity in estrogenized animals was retained for up to 6 weeks. Smooth **muscle** cells became atrophic in castrated animals. This effect was attenuated in estrogen-treated animals. There was no indication for enhanced collagen synthesis by smooth **muscle** cells. **Actin** and desmin-immunoreactivity were only slightly altered in experimental animals and showed a changed distribution pattern. Prostatic smooth **muscle** cells respond less markedly to hormonal alterations than do the fibroblasts.
CC Microscopy - Histology and histochemistry 01056
Cytology - Animal 02506
Cytology - Human 02508
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Sterols and steroids 10067
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Anatomy 15502
Urinary system - Physiology and biochemistry 15504
Urinary system - Pathology 15506
Reproductive system - Anatomy 16502
Reproductive system - Physiology and biochemistry 16504
Reproductive system - Pathology 16506
Endocrine - Gonads and placenta 17006
Muscle - Physiology and biochemistry 17504
Gerontology - 24500
IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Geriatrics (Human Medicine, Medical Sciences); Metabolism; Muscular System (Movement and Support); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis); Urology (Human Medicine, Medical Sciences)

IT Chemicals & Biochemicals
ACTIN

IT Miscellaneous Descriptors
ACTIN; BENIGN PROSTATIC HYPERPLASIA; COLLAGEN; DESMIN;
 ELDERLY; PROSTATIC BINDING PROTEIN; PROSTATIC SMOOTH **MUSCLE**
 CELLS

ORGN Classifier
 Hominidae 86215
 Super Taxa
 Primates; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 human
 Taxa Notes
 Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGN Classifier
 Muridae 86375
 Super Taxa
 Rodentia; Mammalia; Vertebrata; Chordata; Animalia
 Organism Name
 Muridae
 Taxa Notes
 Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
 Rodents, Vertebrates

RN 132579-20-5 (**ACTIN**)

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DN PREV199395056214
TI Estrogen-induced morphological and immunohistochemical changes in stroma
and epithelium of rat ventral prostate.
AU Zhao, G. W.; Holterhus, P. M.; Dammshaeuser, I.; Hoffbauer, G.; Aum Mueller,
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Muscle - Physiology and biochemistry 17504
Gerontology - 24500
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(Human Medicine, Medical Sciences); Metabolism; Muscular System
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IT Chemicals & Biochemicals
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 Taxa Notes
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ORGN Classifier
 Muridae 86375
 Super Taxa
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ED Entered STN: 9 Feb 1993
Last Updated on STN: 10 Feb 1993
AB Prostatic smooth **muscle** cells have been regarded to play a major pathogenetic role during the development of benign prostatic hyperplasia (BPH) in elderly men. Altered hormonal signals (increased estrogen) have been made responsible for the "metabolic" transformation of prostatic smooth **muscle** cells, which were thought to produce increased amounts of connective tissue fibers observed in BPH. In order to find out the role of metabolically "activated" smooth **muscle** cells, hormone stimulation experiments were performed in male rats. The effects of androgen deprivation and estrogen stimulation were recorded by semiquantitative analysis of intermediate and **myofilament proteins** in stromal smooth **muscle** cells. In castrated or estrogen-treated or estrogen-treated and castrated animals, the reduction of the glandular lumen is the most obvious morphological alteration, accompanied by an increase in connective tissue. Regressive changes occurred most rapidly in castrated animals (already within the first week), slower in castrated estrogen-treated animals and still slower in normal estrogen-treated animals. Regression of the epithelium was accompanied by a marked decrease in immunoreactivity for prostatic binding protein (PBP) in castrated animals, while PBP immunoreactivity in estrogenized animals was retained for up to 6 weeks. Smooth **muscle** cells became atrophic in castrated animals. This effect was attenuated in estrogen-treated animals. There was no indication for enhanced collagen synthesis by smooth **muscle** cells. **Actin** and desmin-immunoreactivity were only slightly altered in experimental animals and showed a changed distribution pattern. Prostatic smooth **muscle** cells respond less markedly to hormonal alterations than do the fibroblasts.
CC Microscopy - Histology and histochemistry 01056
Cytology - Animal 02506
Cytology - Human 02508
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Sterols and steroids 10067
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Anatomy 15502
Urinary system - Physiology and biochemistry 15504
Urinary system - Pathology 15506
Reproductive system - Anatomy 16502
Reproductive system - Physiology and biochemistry 16504
Reproductive system - Pathology 16506
Endocrine - Gonads and placenta 17006
Muscle - Physiology and biochemistry 17504
Gerontology - 24500
IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Geriatrics (Human Medicine, Medical Sciences); Metabolism; Muscular System (Movement and Support); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis); Urology (Human Medicine, Medical Sciences)

IT Chemicals & Biochemicals

ACTIN

IT Miscellaneous Descriptors

ACTIN; BENIGN PROSTATIC HYPERPLASIA; COLLAGEN; DESMIN;
ELDERLY; PROSTATIC BINDING PROTEIN; PROSTATIC SMOOTH **MUSCLE**
CELLS

ORGN Classifier

Hominidae 86215

Super Taxa

Primates; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

human

Taxa Notes

Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

Muridae

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates

RN 132579-20-5 (**ACTIN**)

on STN
 AN 95289839 EMBASE
 DN 1995289839
 TI Troponin C - Troponin I interactions and molecular signalling in cardiac myofilaments.
 AU Solaro R.J.; Lab M.; Landesberg A.; Burkhoff D.; Ter Keurs H.
 CS Department of Physiology/Biophysics, College of Medicine, University of Illinois, 901 South Wolcott, Chicago, IL 60612-7342, United States
 SO Advances in Experimental Medicine and Biology, (1995) Vol. 382, pp. 109-115.
 ISSN: 0065-2598 CODEN: AEMBAP
 CY United States
 DT Journal; Conference Article
 FS 002 Physiology
 018 Cardiovascular Diseases and Cardiovascular Surgery
 030 Pharmacology
 037 Drug Literature Index
 LA English
 SL English
 ED Entered STN: 951017
 Last Updated on STN: 951017
 AB This chapter describes a current perception of the molecular interactions regulating myofilament activity in heart cells. The focus is on the interaction between troponin-C (TnC), the Ca²⁺-receptor and troponin I (TnI), an inhibitory protein. It is this interaction that appears to form a molecular switch that turns on the thin filament. It will be seen that control of the **actin-myosin** reaction is not only through Ca²⁺-binding to TnC, but also through steric, cooperative and allosteric processes involving all of the main **myofilament proteins** -**actin**, myosin, tropomyosin (Tm), troponin T (TnT), TnC, and TnI. The process is modulated by covalent and non-covalent mechanisms. The process is altered in diverse myopathies and pathologies of the heart and is a target for pharmacological manipulation by a new class of inotropic agents, the 'Ca²⁺-sensitizers'.
 CT Medical Descriptors:
 ***actin myosin interaction**
 *cardiomyopathy: DT, drug therapy
 *cardiomyopathy: ET, etiology
 *molecular interaction
 allosterism
 calcium binding
 conference paper
 heart muscle cell
 myofilament
 priority journal
 protein phosphorylation
 signal transduction
 Drug Descriptors:
 *calcium ion
 *inotropic agent: DT, drug therapy
 *inotropic agent: PD, pharmacology
 *troponin c
 *troponin i
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 myosin
 protein kinase c
 tropomyosin
 troponin t
 RN (calcium ion) 14127-61-8; (troponin c) 56094-11-2; (troponin i) 77108-40-8; (protein kinase c) 141436-78-4; (tropomyosin) 72067-79-9; (troponin t) 60304-72-5

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on STN
AN 1998056030 EMBASE
TI Breakdown and release of **myofilament proteins** during ischemia and ischemia/reperfusion in rat hearts identification of degradation products and effects on the pCa-force relation.
AU Van Eyk J.E.; Powers F.; Law W.; Larue C.; Hodges R.S.; Solaro R.J.
CS Dr. J.E. Van Eyk, Department of Physiology, Queen's University, Kingston, Ont. K7L 3W6, Canada
SO Circulation Research, (9 Feb 1998) Vol. 82, No. 2, pp. 261-271.
Refs: 56
ISSN: 0009-7330 CODEN: CIRUAL
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
LA English
SL English
ED Entered STN: 19980312
Last Updated on STN: 19980312
AB Our objective in experiments reported here was to identify **myofilament proteins** of rat hearts either lost or degraded by cardiac ischemia (15- or 60-minute duration) with and without 45 minutes of reperfusion. We correlated these changes with alterations in myofilament sensitivity to Ca²⁺ and maximum force generation. Protein degradation and loss were assessed by high- performance liquid chromatography, SDS-PAGE, Western blotting analysis, and amino acid sequencing. Compared with nonischemic control hearts, bundles of skinned fibers from hearts subjected to ischemia alone demonstrated a decrease in maximum force generation and an increase in sensitivity to Ca²⁺. These changes in function were increased with the duration of the ischemia and with reperfusion. With increasing duration of ischemia, there was an increased loss and degradation of myofibrillar α - **actinin** and troponin I (TnI) at its C-terminus. α - **Actinin** and TnI were most susceptible to ischemia, but with 60 minutes of ischemia/reperfusion, there was also degradation of myosin light chain-1 (MLC1) involving a clip of residues 1 to 19. The MLC1 degradation product was detected in the reperfusion effluent (along with troponin T, tropomyosin, and α - **actinin**) but not in the tissue with 60 minutes of ischemia with no reperfusion. Moreover, with ischemia the following proteins became associated with the myofibrils: GAPDH and proteins of the mitochondrial ATP synthase complex. Our results provide new evidence regarding the mechanism by which ischemia/reperfusion causes myocardial injury and support the hypothesis that an important element in the injury is altered activity and structure of the myofilaments.
CT Medical Descriptors:
 *heart muscle ischemia: ET, etiology
 *heart muscle reperfusion
 *reperfusion injury: ET, etiology
 pathophysiology
 protein secretion
 myofilament
 protein degradation
 calcium cell level
 nonhuman
 rat
 animal tissue
 article
 priority journal
Drug Descriptors:
 troponin i: EC, endogenous compound
 tropomyosin: EC, endogenous compound
 alpha actin: EC, endogenous compound

RN (troponin i) 77108-40-8; (tropomyosin) 72067-79-9

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